Sudan University of Sciences& Technology
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Estimation of Kidney size in Ischemic Heart Disease Patients Using Ultrasonography

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بإستخدام التصوير بالموجات فوق الصوتية

A Thesis Submitted for Partial Fulfillment of the Requirment of Master Degree in Medical Diagnostic Ultrasound

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الآية

قال الله تعالى:

(فَتَعَالَى اللَّهُ الْمَلِكُ الْحَقُّ وَلَا تَعْجَلْ بِالْقُرْآنِ مِن قَبْلِ أَن يُقْضَى إِلَيْكَ وَحْيُهُ وَقُل رَّبِّ زِدْنِي عِلْمًا)

صدق الله العظيم

سورة طه الأيه (114)

Dedication

I dedicate this research to my family: parents who were the biggest supporter to me all the way, father mother sisters and brothers who were encouraged me to finish this study.

To my friends and colleagues for their encouragement and emotional support during my study.

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Acknowledgment

My thanks and appreciation to Dr Muna Ahmed Ali for persevering with me as my advisor throughout the time it took me to complete this study. The inspiration for doing this study came from the advanced degree program she had in Sudan University of Science and Technology - Collage of Graduate. The program was one of the most important and formative experiences in my life. I am grateful as well as to Dr. Mohammed Mohammed Omer and Dr. Al Safi Ballah for coordinating and overseeing the administrative concerns that made it possible for me to complete my degree from a geographical distance of 1608.80 mi`les.

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Abstract

Kidney function is affectable by ischemic heart disease and labratory test for renal function test is measuring urea and creatinine level in the blood . the use of ultrasonography give a real time image for shape , size and pathology of the kidney. The purpose of this study was to assess the effect of ischemic heart disease on the renal size using ultrasound and compare the result with blood test (urea and creatinine level) , to measure this effect with age , gender and some ischemic heart disease risk factor (diabetic , hypertention) in order to reduse the mortality rate of ischemic heart disease . ultrasound examination were performed by an expert radiologist in abdominal ultrasound for 58 known ishemic heart disease (diagnosed by cardiologist) 38 male – 20 female. blood test (urea – creatinine) also done. 26 patients are having diabetes millitus and 26 patients having hypertention , 16 patients have both diabetes and hypertension , 22 patients niether diabetic nor hypertensive. The result showed that The Right kidney length and Left kidney length decreases as age increase, while urea and creatinine increases as age increases and there are statistically significant differences between male and female in (Left kidney width), there are statistically significant differences between diabetics and non-diabetics in (Left kidney length and Urea), there are statistically significant differences between hypertensive and non-hypertensive in (Left kidney length) and there are statistically significant differences between both diabetic and hypertensive group and non-diabetic nor hypertensive group in (Left kidney length

and urea).The study recommend for Promote public awareness in developing countries about the nature of ischemic heart disease and early signs for this disease and kidney disease along with patient with history of hypertension and diabetes and control of these risk factors .

المستخلص

يمكن أن تتأثر وظائف الكلى بمرض إحتشاء عضلة القلب، فحص المختبر لوظائف الكلى يقيس نسبة البولينا في الدم ومستوى الكرياتينين في الدم. استخدام الموجات فوق الصوتية يعطي صورة حقيقية لشكل وحجم وأمراض الكلى. هدفت الدراسة إلى تقييم تأثير مرض نقص تروية القلب على حجم الكلى باستخدام الموجات فوق الصوتية ومقارنة النتيجة مع فحص الدم (نسبة البولينا ومستوى الكرياتينين)، مع اعتبار العمر، والجنس، وبعض العوامل الأخرى (مرض السكري، ارتفاع ضغط الدم) لتقليل من معدل وفيات أمراض إحتشاء عضلة القلب وأجرى الفحص بالموجات فوق الصوتية عن طريق اخصائي الاشعة لعدد 58 مريض متابع (تم تشخيصها من قبل طبيب أخصائي بأمراض القلب) منهم 38 ذكور - 20 اناث كما تم فحص (نسبة البولينا في الدم و الكرياتينين) لعدد 26 مريض سكري وضغط و 26 مريض ضغط فقط، 16 سكري فقط، 22 سليماً من السكري والضغط. وأظهرت النتيجة أن طول الكلية يتناقص مع زيادة العمر، في حين ان نسبة البولينا ومستوى الكرياتينين يزيد مع زيادة العمر، وهناك فروق ذات دلالة إحصائية بين الذكور

والإناث في (عرض الكلية اليسرى) كما أن هناك فروق ذات دلالة إحصائية بين مرضى السكري وغير المصابين بالسكري في (طول الكلية اليسرى ونسبة البولينا)، وهناك فروق ذات دلالة إحصائية بين من الذين يعانون من ارتفاع ضغط الدم وعدمه في (طول الكلية اليسرى). أيضاً توجد فروق ذات دلالة إحصائية بين كل من مجموعة مرضى السكري وارتفاع ضغط الدم والسليمين من المرضين السكر وضغط الدم (طول الكلية اليسرى ونسبة البولينا) . أوصيت الدراسة بتعزيز الوعي العام في البلدان النامية حول طبيعة مرض احتشاء عضلة القلب والعلامات المبكرة لهذا المرض وأمراض الكلى جنبا إلى جنب مع المريض مع التاريخ من ارتفاع ضغط الدم ومرض السكري والسيطرة على عوامل الخطر .

كما اوصينا بتطوير يرامج الصحة والتعليم الطبى الرئيسية المتاحة على اساس سنوى لتوفير التثقيف الصحى فى مراكز الرعاية الصحية الأولية وكذلك اوصينا باستخدام الموجات فوق الصوتيه كأداة لفحص المرضى الذين يعانون أمراض نقص تروية القلب مع كل زيارة متابعة .

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List of Abbreviations :

Abbreviation	Full Meaning
IHD	Ischemic heart didease
Pmp	Per milion population
GFR	Glumerulo filtration rate
US	Ultrasound
ADH	Antidiuretic hormone
Co2	Carbone dioxide
NaCl	Sodium chloride
ESRD	End stage renal disease
ACS	Acute coronary syndrome
ST	Period of ventricular depolarization on ECG
STEMI	ST elavation myocardial infarction
CAD	Coronary artery disease
PCI	Percutaneous coronary intervention
CABG	Coronary artery bypas surgery
MCG	Multi function cardiogram
ECG	Electrocardiography
CPT	Current procedural terminology
ECG	Electrocardiography
MRI	Magnetic resonance imaging
CKD	Chronic kidney diseas
CVD	Cardiovascular disease
GFR	Glumerulo filtration rate
NKF-K	Natinal kidney foundation-kidney
DOQI	Diseas outcomes quality intensive
ATP	Adult treatment panel
IDDM	Insuline dependent diabetic millitus
NIDDM	Non insuline dependent diabetic millitus
ACE	Angiotensin converting enzyme
USD	United state dollar
DM	Diabetic millitus
HTN	Hypertention
JINC-7	The seventh report of the joint international committee

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Chapter One

1

Introduction

1.1. Background

Ischemic heart disease *(IHD)* is estimated to be the leading cause of mortality in the world and in high-income countries it is also the leading cause of premature mortality and disability. Each year *IHD* kills an estimated 7 million people representing 13% of all male deaths and 12% of all female deaths (Christopher J. et.al, 2001). *IHD* remain the major cause of premature mortality and morbidity in most of the world (Leon ,et al, 1996).

Chronic kidney disease is at least 3–4 times more frequent in Africa than in developed countries. Hypertension affects < 25% of the adult population and is the cause of chronic kidney failure in 21% of patients on renal replacement therapy in the South African Registry. The prevalence of diabetic nephropathy is estimated to be 14%–16% in South Africa, 23.8% in Zambia, 12.4% in Egypt, 9% in Sudan, and 6.1% in Ethiopia. The current dialysis treatment rate ranges from 70 per million populations (pmp) in South Africa to, 20 pmp in the most of sub-Saharan Africa. The transplant rate in Africa averages 4 pmp and is 9.2 pmp in South Africa. Screening for kidney disease in high-risk populations, eg, patients with hypertension and diabetes mellitus and a family history of kidney disease, should be instituted as the first step in kidney disease prevention in developing countries (Ethn Dis. 2009).

Although in cross-sectional surveys kidney size is directly related to function, the longitudinal relationship between form and function is inverted. Since the rate of change in GFR determines kidney atrophy, we conclude that kidney size is a determinant of renal prognosis (Shathabish et.al, 2009).

The heart can be damaged very early in the course of kidney failure and a number of features of renal disease, particularly high blood pressure, abnormal blood fats (lipids), abnormal levels of calcium and phosphate and the presence of diabetes are associated with an increased cardiovascular mortality rate. This

is why early diagnosis of renal impairment and early management of all factors, which contribute to heart disease , is so important .(. Päivänsalo MJ1,.. ET AL 1998).

Because Ultrasound (US) is one of the most widely used imaging technologies in medicine. It is portable, free of radiation risk, and relatively inexpensive (Vincent chan and Anahi perlas (basics of ultrasound imaging))

The researcher studied the effect on the kidney by screening the kidney size using ultrasound and compare US result to lap test.

1.2. Problem of study

- The *Ischemic heart disease* has major side effect to the renal volume, which can be assessed more precisely using abdominal ultrasound.

1.3. Objectives

1.3.1. General objectives:

This study aims to determine the relation between renal volume and Ischemic heart disease , using Ultrasound.

1.3.2. Specific objectives:

- To measure the renal volume in both male and female.
- To identify the risk factors of Ischemic heart disease including: diabetes mellitus, hypertension.
- To measure urea and creatinine levels in the blood.

1.4. Significant of the study:

To detect if there is effect of Ischemic heart disease in renal volume and advise for management and preserving of risk factor of Ischemic heart disease which that affect the renal volume.

Chapter Two

2. Literature Review

2.1. Kidney Anatomy

The **kidneys** are bean-shaped organs that serve several essential regulatory roles in vertebrates. They remove excess organic molecules from the blood, and it is by this action that their best-known function is performed: the removal of waste products of metabolism. Kidneys are essential to the urinary system and also serve homeostatic functions such as the regulation of electrolytes, maintenance of acid–base balance, and regulation of blood pressure (via maintaining the salt and water balance). They serve the body as a natural filter of the blood, and remove water-soluble wastes which are diverted to the bladder. In producing urine, the kidneys excrete wastes such as urea and ammonium. They are also responsible for the reabsorption of water, glucose, and amino acids. The kidneys also produce hormones including calcitriol, erythropoietin, and the enzyme renin, the last of which indirectly acts on the kidney in negative feedback. (Maton ,… et al 1993).

Located at the rear of the abdominal cavity in the retroperitoneal space, the kidneys receive blood from the paired renal arteries, and drain into the paired renal veins. Each kidney excretes urine into a ureter which empties into the bladder (Maton ,… et al 1993).

Renal physiology is the study of kidney function, while nephrology is the medical specialty concerned with kidney diseases. Diseases of the kidney are diverse, but individuals with kidney disease frequently display characteristic clinical features. Common clinical conditions involving the kidney

include the nephritic and nephrotic syndromes, renal cysts, acute kidney injury, chronic kidney disease, urinary tract infection, nephrolithiasis, and urinary tract obstruction.[1] Various cancers of the kidney exist. The most common adult renal cancer is renal cell carcinoma. Cancers, cysts, and some other renal conditions can be managed with removal of the kidney. This is known as nephrectomy. When renal function, measured by the glomerular filtration rate, is persistently poor, dialysis and kidney transplantation may be treatment options. Although they are not normally , harmful , kidney stone can be painful .
(Maton ,... et al 1993).

2.1.1. Development

The mammalian kidney develops from intermediate mesoderm. Kidney development, also called *nephrogenesis*, proceeds through a series of three successive phases, each marked by the development of a more advanced pair of kidney: the pronephros , mosonephros , and metanephros .
(Bruce M. Carlson 2004).

2.1.2. Location

In humans, the kidneys are located in the abdominal cavity, one on each side of the spine, and lie in a retroperitoneal position at a slightly oblique angle.[2] The asymmetry within the abdominal cavity, caused by the position of the liver, typically results in the right kidney being slightly lower and smaller than the left, and being placed slightly more to the middle than the left kidney.[3][4][5] The left kidney is approximately at the vertebral level T12 to L3,[6] and the right

is slightly lower. The right kidney sits just below the diaphragm and posterior to the liver. The left sits below the diaphragm and posterior to the spleen. On top of each kidney is an adrenal gland. The upper parts of the kidneys are partially protected by the 11th and 12th ribs. Each kidney, with its adrenal gland, is surrounded by two layers of fat: the perirenal and pararenal fat) and the renal fascia. In adult males, the kidney weighs between 125 and 170 grams. In females the weight of the kidney is between 115 and 155 grams (Encyclopedia Britannica 2010).

2.1.3. Structure

The kidney has a bean-shaped structure having a convex and a concave border. A recessed area on the concave border is the renal hilum, where the renal artery enters the kidney and the renal vein and ureter leave. The kidney is surrounded by tough fibrous tissue, the renal capsule, which is itself surrounded by perirenal fat (adipose capsule), renal fascia, and pararenal fat (paranephric body). The anterior (front) surface of these tissues is the peritoneum, while the posterior (rear) surface is the transversalis fascia.

The superior pole of the right kidney is adjacent to the liver. For the left kidney, it's next to the spleen. Both, therefore, move down upon inhalation.

The kidney is approximately 11–14 cm (4.3–5.5 in) in length, 6 cm (2.4 in) wide and 4 cm (1.6 in) thick. (Maton,… et al 1993).

The substance, or parenchyma, of the kidney is divided into two major structures: the outer renal cortex and the inner

renal medulla. Grossly, these structures take the shape of eight to 18 cone-shaped renal lobes, each containing renal cortex surrounding a portion of medulla called a renal pyramid (of Malpighi).[7] Between the renal pyramids are projections of cortex called renal columns (or Bertin columns). Nephrons, the urine-producing functional structures of the kidney, span the cortex and medulla. The initial filtering portion of a nephron is the renal corpuscle which is located in the cortex. This is followed by a renal tubule that passes from the cortex deep into the medullary pyramids. Part of the renal cortex, a medullary ray is a collection of renal tubules that drain into a single collecting duct.

The tip, or papilla, of each pyramid empties urine into a minor calyx; minor calyces empty into major calyces, and major calyces empty into the renal pelvis. This becomes the ureter. At the hilum, the ureter and renal vein exit the kidney and the renal artery enters. Hilar fat snd lymphatic tissue with lymph nodes surrounds these structures. The hilar fat is contiguous with a fat-filled cavity called the renal sinus. The renal sinus collectively contains the renal pelvis and calyces and separates these structures from the renal medullary tissue. (Daniel Zohary and Maria 2012)

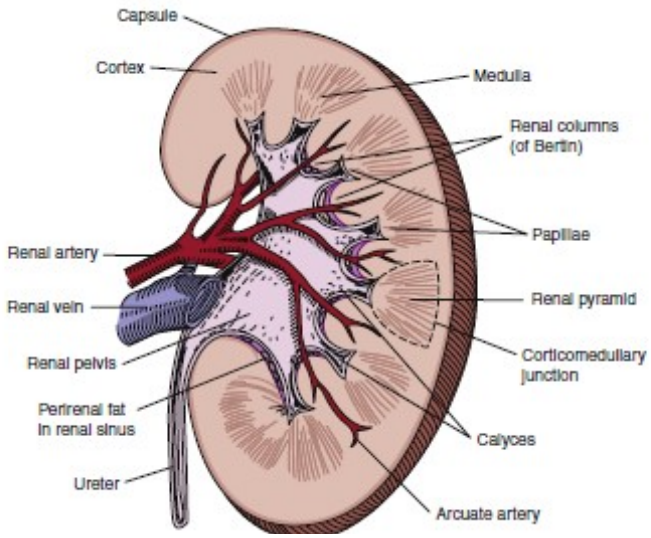

FIGURE 2-1 Diagram of the cut surface of a bisected kidney, depicting important anatomic structures (brener).

2.1.4. Blood Supply

The renal circulation supplies the blood to the kidneys via the renal arteries, left and right, which branch directly from the abdominal aorta. Despite their relatively small size, the kidneys receive approximately 20% of the cardiac output .

Each renal artery branches into segmental arteries, dividing further into interlobar arteries, which penetrate the renal capsule and extend through the renal columns between the renal pyramids. The interlobar arteries then supply blood to the arcuate arteries that run through the boundary of the cortex and the medulla. Each arcuate artery supplies several interlobular arteries that feed into the afferent arterioles that supply the glomeruli.

The medullary **interstitium** is the functional space in the kidney beneath the individual filters (glomeruli), which are rich in blood vessels. The interstitium absorbs fluid recovered from urine. Various conditions can lead to scarring and congestion of this area, which can cause kidney dysfunction and failure.

After filtration occurs, the blood moves through a small network of venules that converge into interlobular veins. As with the arteriole distribution, the veins follow the same pattern: the interlobular provide blood to the arcuate veins then back to the interlobar veins, which come to form the renal vein exiting the kidney for transfusion for blood (aytac ,….. et al 2003).

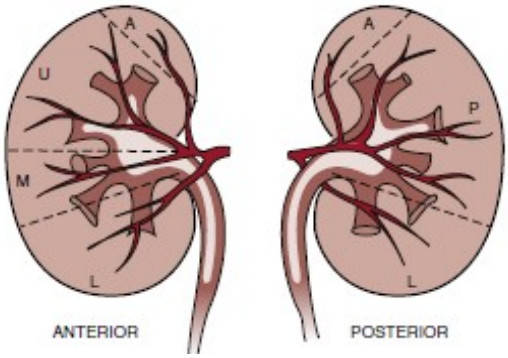

FIGURE 2-2 Diagram of the vascular supply of the human kidney (brener).

2.1.5. Innervation

The kidney and nervous system communicate via the renal plexus, whose fibers course along the renal arteries to reach each kidney.Input from the sympathetic nervous system triggers vasoconstriction in the kidney, thereby reducing renal blood flow.The kidney also receives input from the parasympathetic nervous system, by way of the renal branches of the vagus nerve (cranial nerve X); the function of this is yet unclear.Sensory input from the kidney travels to the T10-11 levels of the spinal cord and is sensed in the corresponding dermatome. Thus, pain in the flank region may be referred from corresponding kidney (Dorland's 2012).

2.2 Function

The kidney participates in whole-body homeostasis, regulating acid-base balance, electrolyte concentrations, extracellular fluid volume, and blood pressure. The kidney accomplishes these homeostatic functions both independently and in concert with other organs, particularly those of the endocrine system. Various endocrine hormones coordinate these endocrine functions; these include renin, angiotensin II, aldosterone, antidiuretic hormone, and atrial natriuretic peptide, among others . (Stevens LA ….et al 2006).

Many of the kidney's functions are accomplished by relatively simple mechanisms of filtration, reabsorption, and secretion, which take place in the nephron. Filtration, which takes place at the renal corpuscle, is the process by which cells and large proteins are filtered from the blood to make an ultrafiltrate that eventually becomes urine. The kidney generates 180 liters of filtrate a day, while reabsorbing a large percentage, allowing for the generation of only approximately 2 liters of urine. Reabsorption is the transport of molecules from this ultrafiltrate and into the blood. Secretion is the reverse process, in which molecules are transported in the opposite direction, from the blood into the urine (Stevens LA ….et al 2006).

2.2.1 Excretion of wastes

The kidneys excrete a variety of waste products produced by metabolism into the urine. These include the nitrogenous wastes urea, from protein catabolism, and uric acid, from nucleic acid metabolism. The ability of mammals and some birds to concentrate wastes into a volume of urine much

smaller than the volume of blood from which the wastes were extracted is dependent on an elaborate countercurrent multiplication mechanism. This requires several independent nephron characteristics to operate: a tight hairpin configuration of the tubules, water and ion permeability in the descending limb of the loop, water impermeability in the ascending loop, and active ion transport out of most of the ascending limb. In addition, passive countercurrent exchange by the vessels carrying the blood supply to the nephron is essential for enabling this function . (Stevens LA ….et al 2006).

2.2.2. Reabsorption of vital nutrients

Glucose at normal plasma levels is completely reabsorbed in the proximal tubule. The mechanism for this is the Na+/glucose cotransporter. A plasma level of 350 mg/dL will fully saturate the transporters and glucose will be lost in the urine. A plasma glucose level of approximately 160 is sufficient to allow glucosuria, which is an important clinical clue to diabetes mellitus.Amino acids are reabsorbed by sodium dependent transporters in the proximal tubule. Hartnup disease is a deficiency of the tryptophan amino acid transporter, which results in pellagra . (Stevens LA ….et al 2006).

2.2.3 Acid-base homeostasis

Two organ systems, the kidneys and lungs, maintain acid-base homeostasis, which is the maintenance of pH around a relatively stable value. The lungs contribute to acid-base homeostasis by regulating carbon dioxide (CO_2) concentration. The kidneys have two very important roles in maintaining the acid-base balance: to reabsorb and regenerate bicarbonate

from urine, and to excrete hydrogen ions and fixed acids (anions of acids) into urine (Stevens LA ....et al 2006)

2.2.4. Osmolality regulation

Any significant rise in plasma osmolality is detected by the hypothalamus, which communicates directly with the posterior pituitary gland. An increase in osmolality causes the gland to secrete antidiuretic hormone (ADH), resulting in water reabsorption by the kidney and an increase in urine concentration. The two factors work together to return the plasma osmolality to its normal levels.

ADH binds to principal cells in the collecting duct that translocate aquaporins to the membrane, allowing water to leave the normally impermeable membrane and be reabsorbed into the body by the vasa recta, thus increasing the plasma volume of the body. (Stevens LA ….et al 2006)

There are two systems that create a hyperosmotic medulla and thus increase the body plasma volume: Urea recycling and the 'single effect.'

Urea is usually excreted as a waste product from the kidneys. However, when plasma blood volume is low and ADH is released the aquaporins that are opened are also permeable to urea. This allows urea to leave the collecting duct into the medulla creating a hyperosmotic solution that 'attracts' water. Urea can then re-enter the nephron and be excreted or recycled again depending on whether ADH is still present or not. (Stevens LA ....et al 2006)

The 'Single effect' describes the fact that the ascending thick limb of the loop of Henle is not permeable to water but is

permeable to NaCl. This allows for a countercurrent exchange system whereby the medulla becomes increasingly concentrated, but at the same time setting up an osmotic gradient for water to follow should the aquaporins of the collecting duct be opened by ADH (Stevens LA ....et al 2006)).

2.2.5. Blood pressure regulation

Although the kidney cannot directly sense blood, long-term regulation of blood pressure predominantly depends upon the kidney. This primarily occurs through maintenance of the extracellular fluid compartment, the size of which depends on the plasma sodium concentration. Renin is the first in a series of important chemical messengers that make up the renin-angiotensin system. Changes in renin ultimately alter the output of this system, principally the hormones angiotensin II and aldosterone. Each hormone acts via multiple mechanisms, but both increase the kidney's absorption of sodium chloride, thereby expanding the extracellular fluid compartment and raising blood pressure. When renin levels are elevated, the concentrations of angiotensin II and aldosterone increase, leading to increased sodium chloride reabsorption, expansion of the extracellular fluid compartment, and an increase in blood pressure. Conversely, when renin levels are low, angiotensin II and aldosterone levels decrease, contracting the extracellular fluid compartment, and decreasing blood pressure (Stevens LA ….et al 2006) .

2.2.6. Hormone Secretion

The kidneys secrete a variety of hormones, including erythropoietin, and the enzyme renin. Erythropoietin is

released in response to hypoxia (low levels of oxygen at tissue level) in the renal circulation. It stimulates erythropoiesis (production of red blood cells) in the bone marrow. Calcitriol, the activated form of vitamin D, promotes intestinal absorption of calcium and the renal reabsorption of phosphate. Part of the renin–angiotensin–aldosterone system, renin is an enzyme involved in the regulation of aldosterone levels (Stevens LA ….et al 2006).

2.2 Kidney Pathology

- Pyelonephritis (infection of kidney pelvis): Bacteria may infect the kidney, usually causing back pain and fever. A spread of bacteria from an untreated bladder infection is the most common cause of pyelonephritis.
- Chronic renal failure: A permanent partial loss of kidney function. Diabetes and high blood pressure are the most common causes.
- End stage renal disease (ESRD): Complete loss of kidney function, usually due to progressive chronic kidney disease. People with ESRD require regular dialysis for survival.
- Diabetic nephropathy: High blood sugar from diabetes progressively damages the kidneys, eventually causing chronic kidney disease. Protein in the urine (nephrotic syndrome) may also result.
- Hypertensive nephropathy: Kidney damage caused by high blood pressure. Chronic renal failure may eventually result.

Nephrogenic diabetes insipidus: The kidneys lose the ability to concentrate the urine, usually due to a drug reaction. Although

it's rarely dangerous, diabetes insipidus causes constant thirst and frequent urination (Appell, R. (2002).

2.4. Ischemic Heart Disease

2.4.1. Definition

Ischemic heart disease is a condition of recurring chest pain or discomfort that occurs when a part of the heart does not receive enough blood. This condition occurs most often during exertion or excitement, when the heart requires greater blood flow. Ischemic heart disease, also called coronary heart disease, is common in the United States and is a leading cause of death worldwide.

Ischemic heart disease develops when cholesterol particles in the blood begin to accumulate on the walls of the arteries that supply blood to the heart. Eventually, deposits called plaques may form. These deposits narrow the arteries and eventually block the flow of blood. This decrease in blood flow reduces the amount of oxygen supplied to the heart muscle (IHD) (wong ,....et al 2014).

2.4.2. Epidemiology

Coronary heart disease is the most common cause of death in the United Kingdom. In total, 220 000 deaths were attributable to ischaemic heart disease in 2007. It is estimated that the incidence of acute coronary syndrome (ACS) is over 250 000 per year.

Sudden death remains a frequent complication of ACS: approximately

50 per cent of patients with ST elevation myocardial infarction (STEMI) do not survive, with around two-thirds of the deaths

occurring shortly after the onset of symptoms and before admission to hospital. Prior to the development of modern drug regimes and reperfusion strategies, hospital mortality after admission with ACS was 30–40 per cent. After the introduction of coronary care units in the 1960s, outcome was improved, predominantly reflecting better treatment of arrhythmias. Current therapy has improved outcome further for younger patients who present early in the course of their ACS. The last decade has seen a significant fall in the overall 30-day mortality rate. Most patients who die before discharge do so in the first 48 hours after admission, usually due to cardiogenic shock consequent upon extensive left ventricular damage. Most patients who survive to hospital discharge do well, with 90 per cent surviving at least one year. Surviving patients who are at increased risk of early death can be identified by a series of adverse clinical and investigational features, and their prognosis improved by intervention (wong , ….et al 2014).

2.4.3. Pathophysiology

ACSs are caused by an imbalance between myocardial oxygen demand and supply those results in cell death and myocardial necrosis. Primarily, this occurs due to factors affecting the coronary arteries, but may also occur as a result of secondary processes such as hypoxemia or hypotension and factors that increase myocardial oxygen demand. The commonest cause is rupture or erosion of an atherosclerotic plaque that leads to complete occlusion of the artery or partial occlusion with distal embolization of thrombotic material.

Atherosclerosis is a disease of large and medium-sized arteries, affecting predominantly the arterial intima. The precise mechanism responsible for the generation of atherosclerotic arterial disease remains open to debate, but it is likely that arterial endothelial injury is an initiating factor (wong ,....et al 2014).

Risk factor:

Risk factors include: high blood pressure, smoking, diabetes, lack of exercise, obesity, high blood cholesterol, poor diet, and excessive alcohol, among others. Other risks include depression. The underlying mechanism involves atherosclerosis of the arteries of the heart. A number of tests may help with diagnoses including: electrocardiogram, cardiac stress testing, and coronary angiogram among others. Prevention is by eating a healthy diet, regular exercise, maintaining a healthy weight and not smoking. Sometimes medication for diabetes, high cholesterol, or high blood pressure are also used.

There is limited evidence for screening people who are at low risk and do not have symptoms. Treatment involves the same measures as prevention. Additional medications such as aspirin, beta blockers, or nitroglycerin may be recommended. Procedures such as percutaneous coronary intervention (PCI) or coronary artery bypass surgery (CABG) may be used in severe disease.

In those with stable CAD it is unclear if PCI or CABG in addition to the other treatments improve life expectancy or decreases heart attack risk.

In 2013 CAD was the most common cause of death globally, resulting in 8.14 million deaths (16.8%) up from 5.74 million deaths (12%) in 1990. The risk of death from CAD for a given age has decreased between 1980 and 2010 especially in the developed world. The number of cases of CAD for a given age has also decreased between 1990 and 2010.[20] In the United States in 2010 about 20% of those over 65 had CAD, while it was present in 7% of those 45 to 64, and 1.3% of those 18 to 45. Rates are higher among men than women of a given age (wong ,....et al 2014).

1- **Signs and symptoms**

Chest pain that occurs regularly with activity, after eating, or at other predictable times is termed stable angina and is associated with narrowing of the arteries of the heart. Angina that changes in intensity, character or frequency is termed unstable. Unstable angina may precede myocardial infarction. In adults who go to the emergency with an unclear cause of pain, about 30% have pain due to coronary artery disease (wong ,....et al 2014).

Diagnosis

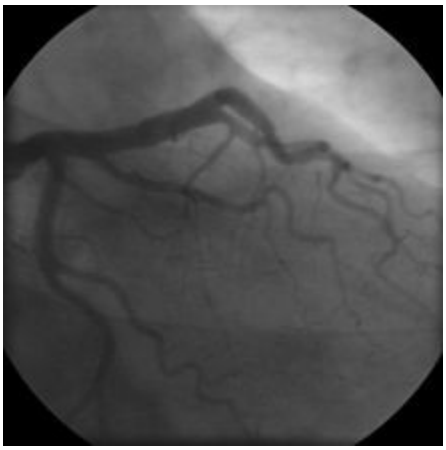

Figure 2-3 Coronary angiogram of a man

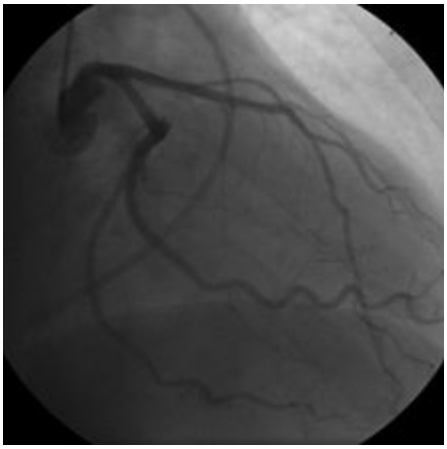

Figure 2-4 Coronary angiogram of a woman

For symptomatic patients, stress echocardiography can be used to make a diagnosis for obstructive coronary artery disease. The use of echocardiography, stress cardiac imaging, and/or advanced non-invasive imaging is not recommended on individuals who are exhibiting no symptoms and are otherwise at low risk for developing coronary disease.

CAD has always been a tough disease to diagnose without the use of invasive or stressful activities. The development of the Multifunction Cardiogram (MCG) has changed the way CAD

is diagnosed. The MCG consists of a 2 lead resting EKG signal is transformed into a mathematical model and compared against tens of thousands of clinical trials to diagnose a patient with an objective severity score, as well as secondary and tertiary results about the patient's condition. The results from MCG tests have been validated in 8 clinical trials[citation needed] which resulted in a database of over 50,000 patients where the system has demonstrated accuracy comparable to coronary angiography (90% overall sensitivity, 85% specificity). This level of accuracy comes from the application of advanced techniques in signal processing and systems analysis combined with a large scale clinical database which allows MCG to provide quantitative, evidence-based results to assist physicians in reaching a diagnosis. The MCG has also been awarded a Category III CPT code by the American Medical Association in the July 2009 CPT update (wong ,....et al 2014).

The diagnosis of "Cardiac Syndrome X" - the rare coronary artery disease that is more common in women, as mentioned, an "exclusion" diagnosis. Therefore, usually the same tests are used as in any patient with the suspicion of coronary artery disease:

- Baseline electrocardiography (ECG).
- Exercise ECG – Stress test.
- Exercise radioisotope test (nuclear stress test, myocardial scintigraphy).
- Echocardiography (including stress echocardiography).
- Coronary angiography.

- Intravascular ultrasound.
- Magnetic resonance imaging (MRI).

The diagnosis of coronary disease underlying particular symptoms depends largely on the nature of the symptoms. The first investigation is an electrocardiogram (ECG/EKG), both for "stable" angina and acute coronary syndrome. An X-ray of the chest and blood tests may be performed (wong ,....et al 2014). There is a high prevalence of CVD in subjects with CKD. The presence of CKD, whether it is manifested by proteinuria (albuminuria) or reduced GFR, appears to be an independent risk factor for CVD outcomes, particularly in higher-risk populations. These findings are consistent with the NKF task force recommendation that patients with CKD should be considered in the highest-risk group for CVD events. The seventh report of the Joint National Committee for the Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC-7) includes CKD as a “compelling” indication, justifying lower target blood pressure and treatment with specific antihypertensive agents.186 Similarly, the recently published “NKF-K/DOQI Clinical Practice Guidelines on Managing Dyslipidemia in Chronic Kidney Disease” recommend that all patients with CKD be included in the highest-risk group, justifying a lower target low-density lipoprotein cholesterol level.53 By contrast, the third report of the Adult Treatment Panel of the National Cholesterol Education Program (ATP-III) does not include CKD in the list of high-risk conditions necessitating more aggressive management.187 We suggest that the National Cholesterol

Education Program and other groups include CKD in the highest-risk group for recommendations for prevention, detection, and treatment of CVD risk factors. In addition, these findings reinforce the recent recommendation from the NKF on the importance of early identification and treatment of CKD and its associated comorbid conditions. We suggest that the routine evaluation of patients with CVD or those at high risk for CVD include measurement of spot urine albumin-to-creatinine ratio or total protein-to-creatinine ratio and estimation of GFR by serum creatinine and prediction equations. Finally, there is an urgent need for additional randomized controlled studies to evaluate potential treatments of CVD in CKD (Williams JG 2011).

2.5. Diabetes mellitus (DM)

2.5.1. Diabetes mellitus definition

Diabetes mellitus (**DM**), commonly referred to as **diabetes**, is a group of metabolic diseases in which there are high blood sugar levels over a prolonged period. Symptoms of high blood sugar include frequent urination, increased thirst, and increased hunger. If left untreated, diabetes can cause many complications. Acute complications include diabetic ketoacidosis and nonketotic hyperosmolar coma. Serious long-term complications include cardiovascular disease, stroke, chronic kidney failure, foot ulcers, and damage to the eyes. Diabetes is due to either the pancreas not producing enough insulin or the cells of the body not responding properly to the insulin produced. There are three main types of diabetes mellitus:

- Type 1 DM results from the pancreas' failure to produce enough insulin. This form was previously referred to as "insulin-dependent diabetes mellitus" (IDDM) or "juvenile diabetes". The cause is unknown.
- Type 2 DM begins with insulin resistance, a condition in which cells fail to respond to insulin properly. As the disease progresses a lack of insulin may also develop. This form was previously referred to as "non-insulin-dependent diabetes mellitus" (NIDDM) or "adult-onset diabetes". The primary cause is excessive body weight and not enough exercise.
- Gestational diabetes, is the third main form and occurs when pregnant women without a previous history of diabetes develop a high blood sugar level.
- Prevention and treatment involve a healthy diet, physical exercise, not using tobacco and being a normal body weight. Blood pressure control and proper foot care are also important for people with the disease. Type 1 diabetes must be managed with insulin injections. Type 2 diabetes may be treated with medications with or without insulin.Insulin and some oral medications can cause low blood sugar. Weight loss surgery in those with obesity is sometimes an effective measure in those with type 2 DM.Gestational diabetes usually resolves after the birth of the baby.
- As of 2014, an estimated 387 million people have diabetes worldwide. With type 2 diabetes making up about 90% of the cases .This represents 8.3% of the adult

population .with equal rates in both women and men. From 2012 to 2014, diabetes is estimated to have resulted in 1.5 to 4.9 million deaths each year. Diabetes at least doubles a person's risk of death. The number of people with diabetes is expected to rise to 592 million by 2035.The global economic cost of diabetes in 2014 was estimated to be $612 billion USD .In the United States, diabetes cost $245 billion in 2012

- The classic symptoms of untreated diabetes are weight loss, polyuria (increased urination), polydipsia (increased thirst), and polyphagia (increased hunger). Symptoms may develop rapidly (weeks or months) in type 1 diabetes, while they usually develop much more slowly and may be subtle or absent in type 2 diabetes.

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2.5.2. **Pathophysioloy**

Insulin is the principal hormone that regulates the uptake of glucose from the blood into most cells of the body, especially liver, muscle, and adipose tissue. Therefore, deficiency of insulin or the insensitivity of its receptors plays a central role in all forms of diabetes mellitus. The body obtains glucose from three main places: the intestinal absorption of food, the

breakdown of glycogen, the storage form of glucose found in the liver, and gluconeogenesis, the generation of glucose from non-carbohydrate substrates in the body. Insulin plays a critical role in balancing glucose levels in the body. Insulin can inhibit the breakdown of glycogen or the process of gluconeogenesis, it can stimulate the transport of glucose into fat and muscle cells, and it can stimulate the storage of glucose in the form of glycogen. Insulin is released into the blood by beta cells (β-cells), found in the islets of Langerhans in the pancreas, in response to rising levels of blood glucose, typically after eating. Insulin is used by about two-thirds of the body's cells to absorb glucose from the blood for use as fuel, for conversion to other needed molecules, or for storage. Lower glucose levels result in decreased insulin release from the beta cells and in the breakdown of glycogen to glucose. This process is mainly controlled by the hormone glucagon, which acts in the opposite manner to insulin. If the amount of insulin available is insufficient, if cells respond poorly to the effects of insulin (insulin insensitivity or insulin resistance), or if the insulin itself is defective, then glucose will not be absorbed properly by the body cells that require it, and it will not be stored appropriately in the liver and muscles. The net effect is persistently high levels of blood glucose, poor protein synthesis, and other metabolic derangements, such as acidosis. When the glucose concentration in the blood remains high over time, the kidneys will reach a threshold of reabsorption, and glucose will be excreted in the urine (glycosuria).This increases the osmotic pressure of the urine

and inhibits reabsorption of water by the kidney, resulting in increased urine production (polyuria) and increased fluid loss. Lost blood volume will be replaced osmotically from water held in body cells and other body compartments, causing dehydration and increased thirst (polydipsia).(Kittell F (2012)

2.6. Hypertention

2.6.1. Definition

Hypertension (**HTN** or **HT**), also known as **high blood pressure** or **arterial hypertension**, is a chronic medical condition in which the blood pressure in the arteries is elevated. Blood pressure is expressed by two measurements, the systolic and diastolic pressures, which are the maximum and minimum pressures, respectively, in the arterial system. The systolic pressure occurs when the left ventricle is most contracted; the diastolic pressure occurs when the left ventricle is most relaxed prior to the next contraction. Normal blood pressure at rest is within the range of 100–140 mmHg systolic and 60–90 mmHg diastolic. Hypertension is present if the blood pressure is persistently at or above 140/90 millimeters mercury (mmHg) for most adults; different criteria apply to children.(lackland 2015)

Hypertension usually does not cause symptoms initially, but sustained hypertension over time is a major risk factor for hypertensive heart disease, coronary artery disease, stroke, aortic aneurysm, peripheral artery disease, and chronic kidney disease. .(lackland 2015)

Hypertension is classified as either primary (essential) hypertension or secondary hypertension. About 90–95% of

cases are categorized as primary hypertension, defined as high blood pressure with no obvious underlying cause .The remaining 5–10% of cases are categorized as secondary hypertension, defined as hypertension due to an identifiable cause, such as chronic kidney disease, narrowing of the aorta or kidney arteries, or an endocrine disorder such as excess aldosterone, cortisol, or catecholamine. Dietary and lifestyle changes can improve blood pressure control and decrease the risk of health complications, although treatment with medication is still often necessary in people for whom lifestyle changes are not enough or not effective. The treatment of moderately high arterial blood pressure (defined as >160/100 mmHg) with an improved life expectancy. (lackland 2015)

The benefits of treatment of blood pressure that is between 140/90 mmHg and 160/100 mmHg are less clear, with some reviews finding no benefit and other reviews finding benefit (lackland 2015)

Hypertension is rarely accompanied by any symptoms, and its identification is usually through screening, or when seeking healthcare for an unrelated problem. Some with high blood pressure report headaches (particularly at the back of the head and in the morning), as well as lightheadedness, vertigo, tinnitus (buzzing or hissing in the ears), altered vision or fainting episodes .These symptoms, however, might be related to associated anxiety rather than the high blood pressure itself .(lackland 2015)

On physical examination, hypertension may be associated with the presence of change in the optic fundus seen by ophthalmoscopy.(lackland 2015)

The severity of the changes typical of hypertensive retinopathy is graded from I–IV; grades I and II may be difficult to differentiate The severity of the retinopathy correlates roughly with the duration and/or the severity of the hypertension (.(lackland 2015)

2.6.2 Pathophysiology

In most people with established essential hypertension, increased resistance to blood flow (total peripheral resistance) accounts for the high pressure while cardiac output remains normal .There is evidence that some younger people with prehypertension or 'borderline hypertension' have high cardiac output, an elevated heart rate and normal peripheral resistance, termed hyperkinetic borderline hypertension .These individuals develop the typical features of established essential hypertension in later life as their cardiac output falls and peripheral resistance rises with age .Whether this pattern is typical of all people who ultimately develop hypertension is disputed .The increased peripheral resistance in established hypertension is mainly attributable to structural narrowing of small arteries and arterioles although a reduction in the number or density of capillaries may also contribute. Whether increased active arteriolar vasoconstriction plays a role in established essential hypertension is unclear .Hypertension is also associated with decreased peripheral venous compliance which may increase venous return, increase cardiac preload and, ultimately, cause diastolic dysfunction. Pulse pressure (the difference between systolic and diastolic blood pressure) is frequently increased in older people with hypertension. This

can mean that systolic pressure is abnormally high, but diastolic pressure may be normal or low — a condition termed isolated systolic hypertension The high pulse pressure in elderly people with hypertension or isolated systolic hypertension is explained by increased arterial stiffness, which typically accompanies aging and may be exacerbated by high blood pressure. Many mechanisms have been proposed to account for the rise in peripheral resistance in hypertension. Most evidence implicates either disturbances in the kidneys' salt and water handling (particularly abnormalities in the intrarenal renin-angiotensin system) and/or abnormalities of the sympathetic nervous system .These mechanisms are not mutually exclusive and it is likely that both contribute to some extent in most cases of essential hypertension. It has also been suggested that endothelial dysfunction and vascular inflammation may also contribute to increased peripheral resistance and vascular damage in hypertension Interleukin 17 has garnered interest for its role in increasing the production of several other immune system chemical signals thought to be involved in hypertension such as tumor necrosis factor alpha, interleukin 1, interleukin 6, and interleukin 8 .(lackland 2015) .

2.7 Previous **studies:**

Päivänsalo MJ[1] et.al investigated a population-based series (1031 subjects, age range 40-60 years) whether the renal size of hypertensive subjects differs from that of control subjects and whether the difference might be due to hypertension itself or risk factors associated with hypertension. The renal measurements were performed by abdominal ultrasound. The

genders were analyzed separately. Hypertensive men had slightly larger kidneys than controls (70.1+/-8.9 cm2 vs. 67.9+/-8.7 cm2, p <0.008)., whereas hypertension, blood pressure or hypertensive medication did not affect renal size. High serum concentrations of uric acid and creatinine were associated with smaller kidney size (p <0.001 and p <0.05, respectively). Alcohol users had slightly larger kidneys than abstainers, but the difference was not significant. Renal size increased with pack years of smoking. Diabetics had 4.8% larger kidneys (p <0.039), but no difference was observed between the subjects with impaired glucose tolerance and those with normal test results. In multivariate analysis, the most significant factors associated with enlarged kidney size were the fasting blood glucose concentration (p < or = 0.0001), smoking (p < or = 0.0001) and atherosclerotic lesions in carotid arteries (p <0.002). The kidneys were also slightly larger in hypertensive women than in control subjects, but the difference was only of borderline significance (p <0.08). Women on hormone replacement therapy had smaller kidneys than other women (p <0.05), but there was no difference in renal measures between premenopausal and postmenopausal women. In multivariate analysis, the most significant factors contributing to large kidney size were blood glucose concentration (p <0.0001) and smoking (p <0.05), while age and serum creatinine concentration were associated with smaller kidney size (p <0.0001 and p <0.0001). We conclude that renal size is related to sex and the subject's height and weight. Smoking, abnormal glucose tolerance, blood uric acid,

creatinine, carotid atherosclerosis and hormone replacement therapy in women were also significant factors for renal size. Hypertensive subjects had larger kidneys than controls, mainly because of their more frequent obesity and abnormal glucose test.(Chrestoph Hasslecher et al. (2001) .

Patients with diabetes who have a family history of cardiovascular disease or hypertension are at greatly increased risk for development of diabetic nephropathy. The changes that occur in the diabetic hypertensive kidney (mesangial matrix expansion, altered charge and size selectivity of the glomerular basement membrane, and significantly increased intraglomerular pressure) are not generally present in the nondiabetic hypertensive kidney. Angiotensin-converting enzyme (ACE) inhibitors and nondihydropyridine calcium blockers are known to attenuate these changes. Patients taking these agents experience a reduction in the proteinuria associated with nephrotic syndrome; this is accompanied by marked reductions in serum cholesterol level, increases in serum albumin level, and reduced morbidity. Other antihypertensive therapies have not been shown to have these effects. Moreover, ACE inhibitors and alpha blockers have been shown to improve insulin resistance in patients with noninsulin-dependent diabetes. For the patient with diabetes, attention must be given to these factors, and blood pressure medication must be carefully selected (Bakris 1993).

Chapter Three

3.1 Methods and Materials:

The study was conducted in Sudan Heart Centre in Khartoum with known ischemic heart disease (IHD) came to ultrasound department for check their kidney all the patients were >40 years (25) in patient and (33) outpatients

3.2 Inclusion criteria

Known IHD adult diabetic patient and hypertension patient.

3.3 Exclusion criteria:

All others heart diseases. (Myocardial infarction MI, Cardiomyopathy …)

3.4 Data collection:

Data was collected from patients who were referred to sudan heart centre considering their age, family history of heart diseases and was recommended to follow up and inpatient cardiac care unit and intensive care unit.

3.5 Ethical consideration:

All results were taken from patients file after verbal agreement from them and also after agreement of the head of sudan heart center and medical records Clerks.

3.6 Equipment selection:

This study was conducted by (HS _ 2000 CINE) includes Transducer options 3-5 MHz 2D Wide Band CURVED Probe

3.7 Technique:

A 3.5-5 MHz probe is typically used to scan the kidney. All the patients were placed in supine position and the probe was placed in the right lower intercostal space in the midaxillary

line in order to scan the right kidney. The liver has been used as “acoustic window” and the probe has been gently rocked (up and down) to scan the entire kidney. Then the patient was instructed to inspire or exhale that allowed the researcher to subtle movement of the kidney dimension longitudinal (long axis) and transverse (short axis) views more obtained.
For the left kidney the patients lied in supine or in the right lateral decubitus position. The probe was placed at the lower intercostal space on the posterior axillary line. The placement will be more cephalad and posterior than when visualizing the right kidney. The probe was gently rocked to scan the entire kidney. Longitudinal and transverse views more obtained.
the sonographic shape of the kidney changed according to the axis the kidney appeared as a football-shaped and are typically 9-12 cm in length and 4-5 cm in width (normally within 2 cm of each other). On transverse view, the kidney appears as C-shaped. The normal kidney appeared as a bright area surrounding it which is made up of Gerota’s fascia and perinephric fat. The periphery of the kidney will appeared grainy gray which is made up of the renal cortex and pyramids. Sometimes individual pyramids in the case The central area of the kidney, the renal sinus, appears in bright (echogenic) and consists of the calyces, renal pelvis and the renal sinus fat. Both kidneys were scanned for comparison and correlation to clinical picture. The ureters were generally not well visualized by ultrasound, but, when distended may appear as a tubular structure extending inferiorly from the kidney .The bladder, when distended with fluid, can be easily

visualized in the lower pelvis as a rather thick walled, fluid-filled structure.(Thompson and Bhatt 2014).

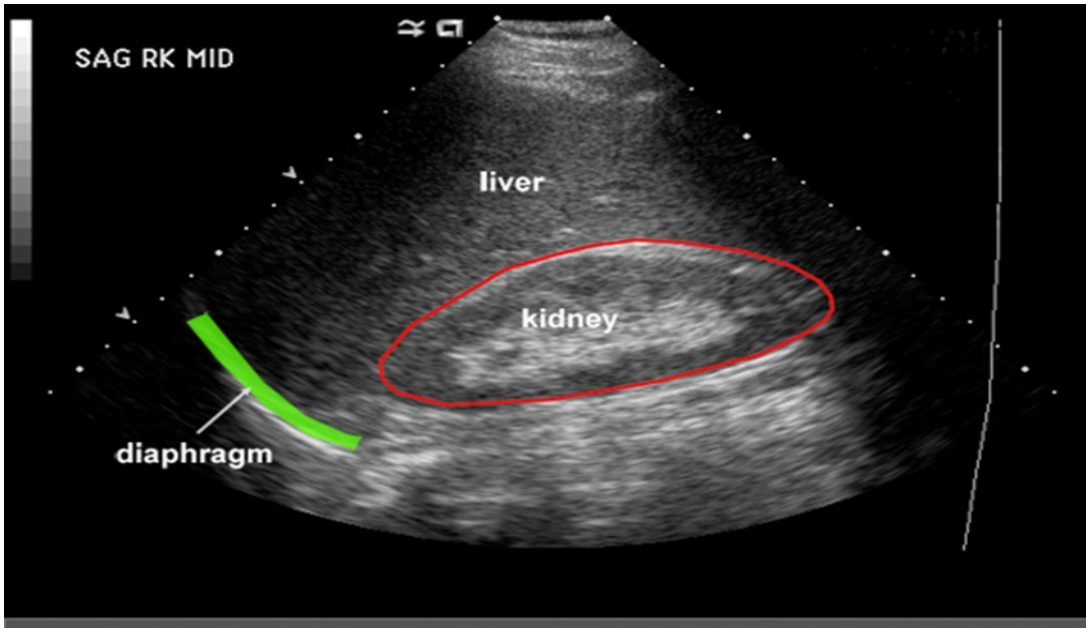

Figure 3-1a longitudinal images of normal right kidney.(Thompson and Bhatt 2014)

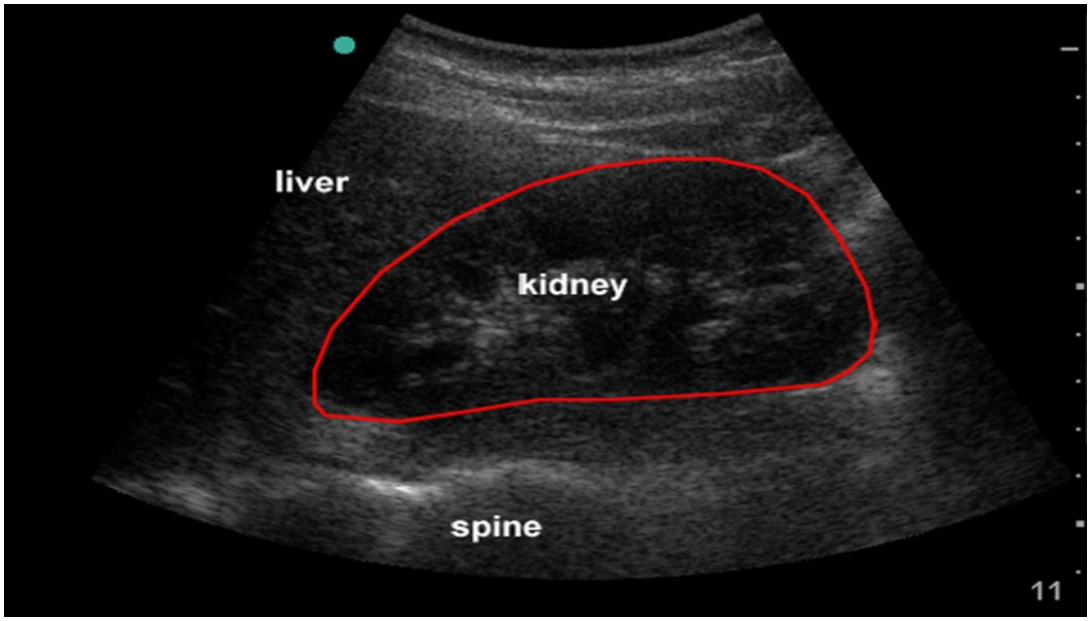

Figure 3-1b longitudinal images of normal right kidney.

Figure 3-1a and 3-1b: Longitudinal images of normal right kidney. (Thompson and Bhatt 2014)

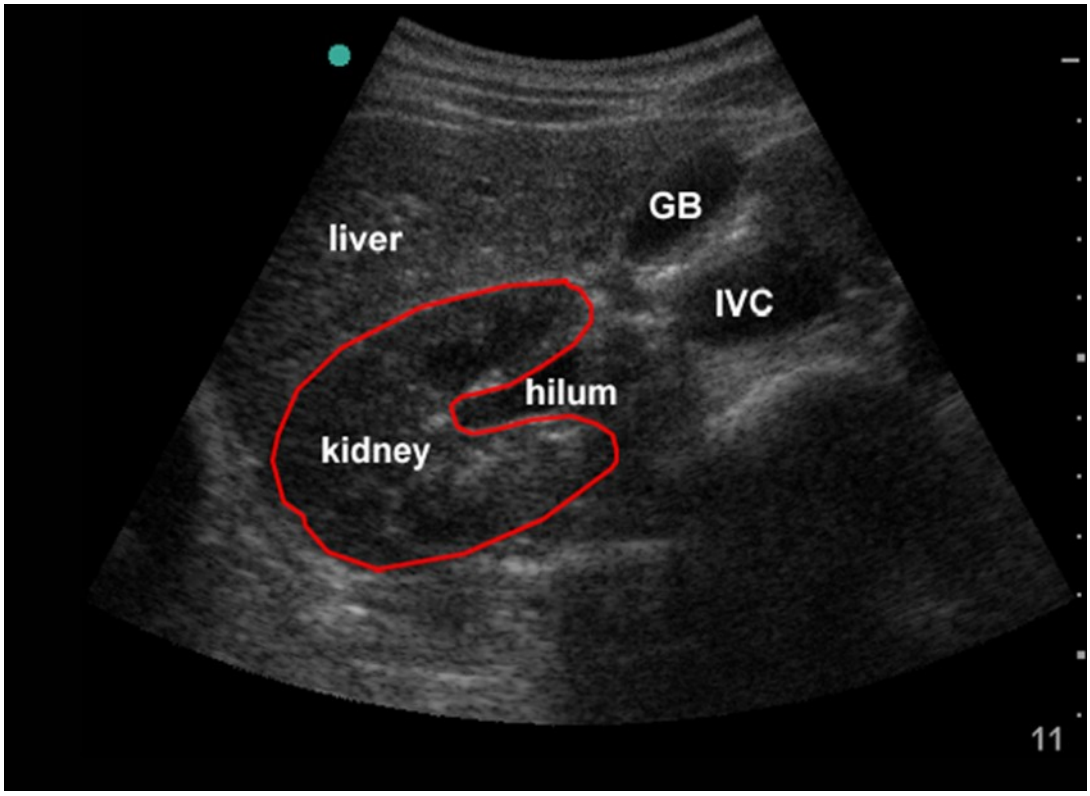

Figure 3-2Transverse image of normal right kidney. (Thompson and Bhatt 2014)

Chapter Four

The results

Table (1) descriptive statistics for (renal size, Creatinine and Urea):

	Mean	Std. Deviation	N
Rt. Kidney Length	90.9348	8.55803	58
Rt. Kidney Width	46.0421	7.13697	58
Lt. kidney Length	89.2283	10.96825	58
Lt. kidney Width	45.6590	9.48750	58
Urea	59.31	40.773	58
Creatinine	1.30394	.649316	58

Table (2) Correlations between age and (renal size, Creatinine, Urea):

		Age	Rt. Kidney Length	Rt. Kidney Width	Lt. kidney Length	Lt. kidney Width	Urea	Creatinine
Age	Pearson Correlation	1	-.413	-.158	-.417	-.063	.283	.277
	Sig. (2-tailed)		.001	.236	.001	.640	.031	.035
	N	58	58	58	58	58	58	58

Table (3) descriptive statistics for (renal size, Creatinine, Urea) with respect to gender:

	Gender	N	Mean	Std. Deviation	Std. Error Mean
Rt. Kidney Length	Male	38	92.4416	7.81089	1.26709
	Female	20	88.0720	9.36712	2.09455
Rt. Kidney Width	Male	38	46.7789	6.56732	1.06536
	Female	20	44.6420	8.10372	1.81205
Lt. kidney Length	Male	38	90.0495	8.87195	1.43922
	Female	20	87.6680	14.27292	3.19152
Lt. kidney Width	Male	38	49.0116	8.52902	1.38359
	Female	20	39.2890	7.94897	1.77744
Urea	Male	38	63.16	44.844	7.275
	Female	20	52.00	31.391	7.019
Creatinine	Male	38	1.42144	.733272	.118952
	Female	20	1.08070	.371061	.082972

Table (4): t-test Equality of (males and females):

		t-test for Equality of Means				
		t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference
Rt. Kidney Length	Equal variances assumed	1.889	56	.064	4.36958	2.31262
	Equal variances not assumed	1.785	33.170	.083	4.36958	2.44799
Rt. Kidney Width	Equal variances assumed	1.086	56	.282	2.13695	1.96853
	Equal variances not assumed	1.017	32.416	.317	2.13695	2.10202
Lt. kidney Length	Equal variances assumed	.783	56	.437	2.38147	3.04034
	Equal variances not assumed	.680	26.941	.502	2.38147	3.50102
Lt. kidney Width	Equal variances assumed	4.222	56	.000	9.72258	2.30305
	Equal variances not assumed	4.316	41.228	.000	9.72258	2.25247
Urea	Equal variances assumed	.990	56	.326	11.158	11.266
	Equal variances not assumed	1.104	51.328	.275	11.158	10.109
Creatinine	Equal variances assumed	1.945	56	.057	.340742	.175148
	Equal variances not assumed	2.349	55.964	.022	.340742	.145031

Table (5) descriptive statistics for (renal size, Creatinine, Urea) in diabetics and non-diabetics:

	DM	N	Mean	Std. Deviation	Std. Error Mean
Rt. Kidney Length	Yes	26	88.8023	8.64061	1.69456
	No	32	92.6675	8.21851	1.45284
Rt. Kidney Width	Yes	26	45.1392	6.80455	1.33448
	No	32	46.7756	7.42144	1.31194
Lt. kidney Length	Yes	26	85.0731	7.88278	1.54594
	No	32	92.6044	12.03509	2.12752
Lt. kidney Width	Yes	26	46.0715	8.89637	1.74472
	No	32	45.3238	10.07083	1.78029
Urea	Yes	26	76.31	49.103	9.630
	No	32	45.50	25.944	4.586
Creatinine	Yes	26	1.46838	.715640	.140349
	No	32	1.17034	.566656	.100171

Table (6): t-test for Equality of Means of two groups (diabetics and non-diabetics):

		t-test for Equality of Means		
		t	df	Sig. (2-tailed)
Rt. Kidney Length	Equal variances assumed	-1.741	56	.087
	Equal variances not assumed	-1.732	52.420	.089
Rt. Kidney Width	Equal variances assumed	-.867	56	.390
	Equal variances not assumed	-.874	55.140	.386
Lt. kidney Length	Equal variances assumed	-2.746	56	.008
	Equal variances not assumed	-2.864	53.785	.006
Lt. kidney Width	Equal variances assumed	.296	56	.768
	Equal variances not assumed	.300	55.575	.765
Urea	Equal variances assumed	3.065	56	.003
	Equal variances not assumed	2.888	36.128	.007
Creatinine	Equal variances assumed	1.771	56	.082
	Equal variances not assumed	1.729	47.101	.090

Table (7) descriptive statistics for (renal size, Creatinine, Urea) in hypertensive and non- hypertensive:

	HTN	N	Mean	Std. Deviation	Std. Error Mean
Rt. Kidney Length	yes	26	92.2985	6.67378	1.30884
	No	32	89.8269	9.79394	1.73134
Rt. Kidney Width	yes	26	46.0715	6.66160	1.30645
	No	32	46.0181	7.60710	1.34476
Lt. kidney Length	yes	26	84.7623	8.38717	1.64486
	No	32	92.8569	11.58243	2.04750
Lt. kidney Width	yes	26	45.9938	7.15853	1.40390
	No	32	45.3869	11.13600	1.96859
Urea	yes	26	63.15	48.607	9.533
	No	32	56.19	33.600	5.940
creatinine	yes	26	1.29746	.767586	.150536
	No	32	1.30921	.547727	.096825

Table (8): t-test for Equality of Means of two groups (hypertensive and non-hypertensive):

Table (9) descriptive statistics for (renal size, Creatinine, Urea) in Both DM. & HTN. Patients and Neither DM. Nor HTN. Ones:

		N	Mean	Std. Deviation
Rt. Kidney Length	Both DM. & HTN.	16	91.5312	6.74357
	Neither DM. Nor HTN.	22	92.2773	8.93437
Rt. Kidney Width	Both DM. & HTN.	16	46.0812	5.87229
	Neither DM. Nor HTN.	22	47.1027	7.26280
Lt. kidney Length	Both DM. & HTN.	16	83.4512	8.12428
	Neither DM. Nor HTN.	22	95.2155	12.56057
Lt. kidney Width	Both DM. & HTN.	16	44.3138	6.34950
	Neither DM. Nor HTN.	22	43.7973	10.73730
Urea	Both DM. & HTN.	16	73.69	57.031
	Neither DM. Nor HTN.	22	45.14	26.899
Creatinine	Both DM. & HTN.	16	1.42662	.851507
	Neither DM. Nor HTN.	22	1.20649	.564941

		t-test for Equality of Means		
		t	df	Sig. (2-tailed)
Rt. Kidney Length	Equal variances assumed	1.096	56	.278
	Equal variances not assumed	1.139	54.489	.260
Rt. Kidney Width	Equal variances assumed	.028	56	.978
	Equal variances not assumed	.028	55.655	.977
Lt. kidney Length	Equal variances assumed	-2.982	56	.004
	Equal variances not assumed	-3.082	55.342	.003
Lt. kidney Width	Equal variances assumed	.240	56	.811
	Equal variances not assumed	.251	53.418	.803
Urea	Equal variances assumed	.644	56	.522
	Equal variances not assumed	.620	42.958	.538
Creatinine	Equal variances assumed	-.068	56	.946
	Equal variances not assumed	-.066	43.904	.948

Table (10): One-sample ANOVA test for Equality of Means of two groups (Both DM. & HTN. Patients and Neither DM. Nor HTN.):

		Sum of Squares	df	Mean Square	F	Sig.
Rt. Kidney Length	Between Groups	534.828	3	178.276	2.645	.058
	Within Groups	3639.846	54	67.405		
	Total	4174.675	57			
Rt. Kidney Width	Between Groups	82.861	3	27.620	.529	.664
	Within Groups	2820.509	54	52.232		
	Total	2903.370	57			
Lt. kidney Length	Between Groups	1403.035	3	467.678	4.630	.006
	Within Groups	5454.214	54	101.004		
	Total	6857.249	57			
Lt. kidney Width	Between Groups	300.599	3	100.200	1.120	.349
	Within Groups	4830.125	54	89.447		
	Total	5130.724	57			
Urea	Between Groups	13909.785	3	4636.595	3.097	.034
	Within Groups	80848.628	54	1497.197		
	Total	94758.414	57			
Creatinine	Between Groups	1.439	3	.480	1.146	.339
	Within Groups	22.593	54	.418		
	Total	24.032	57			

Chapter Five

Chapter 5
Discussion, Conclusion ad Recommendation

5-1 Discussion:

This is a prospective study conducted to estimate the kidney size in ischemic heart disease patients, 58 patients who referred to Sudan heart center in Sudan were assessed .the result of this study showed tables (1, 2) that ischemic heart disease patients had satirically slightly decrease the kidney length (-41%, -42%) for (right and left kidneys) respectively with ($p < 0.05$) as age increase and not significantly the kidney width (-16%, -6%), while (urea and creatinine) significantly increase with ($p < 0.05$). therefore high urea and creatinine associated with smaller kidney size agreed as (Päivänsalo MJ[1] et.al).

From tables (3, 4), ischemic heart disease patients shown slightly smaller kidney size for both male than female (92.4±7.9 mm vs. 88.1±9.4mm), (46.8±6.6mm vs. 44.6±8.1mm), (90±8.9mm vs. 87.7±14.3mm), (49±8.5mm vs. 39.3±7.9mm), (63.2±44.8mm vs.52±31.4mm) respectively for (Rt. K. length, Rt. K. width, Lt. K. length, Rt., Lt. K. width) with no significance other than left kidney width ($p < 0.05$) whereas the results were differ from normal kidney size then are effectible by ischemic heart disease.

Tables (5, 6) shown that diabetes ischemic heart disease patients had no significantly slightly smaller kidney size than non-diabetics (88.8±8.6mm, 92.7±8.2mm), (45.1±6.8mm, 46.8±7.4mm), (85.1±7.9mm, 92.6±12mm), (46.1±9mm,

45.3±010.1mm) for (Rt. K. length, Rt. K. width, Lt. K. length, Rt., Lt. K. width) respectively and vice versa for Urea and Creatinine (76.3±49.1 , 45.5±25.9), (1.5±0.72 , 1.2±0.56) therefore high urea and creatinine associated with smaller kidney size agreed with (Päivänsalo MJ[1] et.al).

Tables (7, 8) shown concerned differences observed between hypertensive ischemic heart disease . and non-hypertensive ischemic heart disease patients in (Rt. Kidney length, Rt. kidney width, Rt., Lt. K. width, Urea and Creatinine) (92.3±6.7mm , 89.8±9.8mm), (46.1±6.7mm , 46±7.6mm) (45.9±7.2mm , 45.4±11.1mm), (63.2±48.6 , 56.2±33.6), (1.2±0.77 , 1.3±0.55) respectively excepting Lt. kidney length (84.7±8.4mm , 92.9±11.1mm) with significant difference ($P < 0.05$).

Tables (9, 10) shown no significant differences between both diabetic & hypertensive. Patients and neither diabetic nor hypertensive in kidney size and lab tests except (Lt. kidney length $P < 0.05$). The changes that occur in diabetic. & hypertensive. Ischemic heart disease Patients are generally not in neither diabetic nor hypertensive.

5.2 conclusion :

Kidney disease and kidney failure, especially as a complication of Ischemic heart disease, are rising globally. Most of kidney ultrasounds for patients show that The Rt. kidney length and Lt. kidney length decreases as age increase, while Urea and Creatinine increases as age increases and there are statistically significant differences between male and female in (Lt. kidney width), there are statistically significant differences between diabetics and non-diabetics in (Lt. kidney length and Urea), there are statistically significant differences between hypertensive and non-hypertensive in (Lt. kidney length) and there are statistically significant differences between both diabetic and hypertensive group and non-diabetic nor hypertensive group in (Lt. kidney length and Urea).

On the other hand, diabetes and hypertension is associated factors that will affect the size of the kidney. Adequate and early detection and aggressive treatment of Ischemic heart disease will reduce the long term effect on kidney. Apparently, this study shows obvious increasing in Creatinine and urea for patient with history of hypertension and diabetes.

5.3 Recommendation:

- Promote public awareness in developing countries about the nature and early signs of ischemic heart disease and kidney disease along with patient with history of hypertension and diabetes and control of these risk factors.
- Make major health and medical education programs available on an annual basis to provide health education in primary health care centers.
- Use ultrasound as screening tool for ischemic heart disease patients with their follow.

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